PROCEEDINGS
OF THE
NATIONAL ACADEMY OF SCIENCES
Volume 6 NOVEMBER 15, 1920 Number 11

HOMOLOGOUS GENES AND LINEAR LINKAGE IN DROSOPHILA VIRILIS

By Alexander Weinstein

Garrison-on-Hudson, New York

Communicated by T. H. Morgan, September 30, 1920

Various species of Drosophila have produced mutations resembling in somatic appearance and in genetic behavior mutations in D. melanogaster.[1] In Sturtevant's case, involving notch in D. funebris, the resemblances to the notch of D. melanogaster are so numerous that it seems extremely probable that the two factors are similar genes in homologous loci. In the other cases, however, the similarities are not sufficient to substantiate such a conclusion.

Unfortunately, in all these instances, proof that the factors would act as allelomorphs if brought together is unavailable because the species studied do not hybridize with each other. However, even without data from hybridization, the homology of factors or of chromosomes in different species might still be detected if it were possible to demonstrate series of linked genes of which the similarly placed members have similar somatic effects. Metz has suggested that the yellow and forked genes in D. virilis are homologous with the similarly named genes in D. melanogaster. This identification, necessarily doubtful because only two factors were involved in each series, was rendered still more questionable by the discovery in D. melanogaster of singed (by Mohr) and inflated (by Weinstein[2]). The location of these factors is shown in figure 1*C*. Inflated resembles vesiculated; and if the two factors were considered homologous, it would be possible (by turning the virilis chromosome round) to homologize forked in virilis with singed in melanogaster. Singed resembles forked in its bristles, but in addition in singed flies the hairs are affected and the females are sterile. The resemblance, therefore, is not so good as that between the two forked characters; still the differences between forked in virilis and singed in melanogaster might be due to modifying factors, or the two genes might be multiple allelomorphs. While, therefore, the identification of forked and vesiculated with singed and inflated is not so

plausible as the identification of the yellow forked series in the two species, it would be impossible in the absence of further evidence to decide which interpretation, if either, is correct.

The balance of proof has swung in favor of the identification of the two yellow forked series by the discovery of the mutant crossveinless in both D. melanogaster (by Bridges) and in D. virilis (by Weinstein). In crossveinless flies of virilis (fig. 2*c*) the posterior crossvein is entirely absent, except that very rarely a small segment in the middle of the crossvein persists; the anterior crossvein is very faint and may be partly missing.

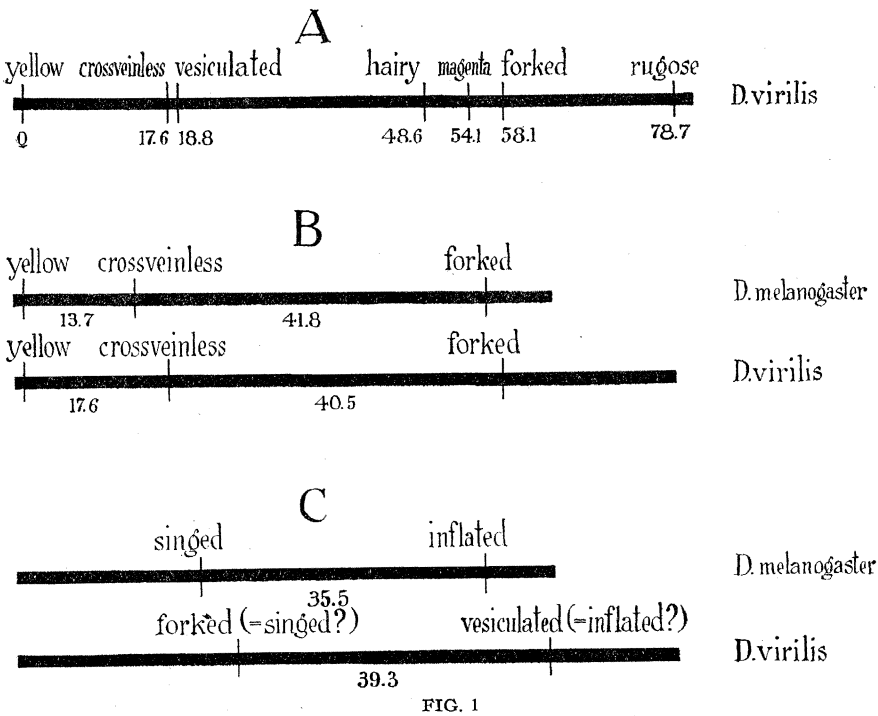

FIG. 1

In crossveinless mutants of melanogaster (fig. 2*b*), the posterior crossvein is missing, the anterior crossvein is very faint and almost entirely absent, and in addition the second longitudinal vein is slightly thickened at its distal end (see Bridges' paper elsewhere in this issue of the PROCEEDINGS, p. 660). Both factors are recessive and sex-linked, and occupy similar positions in the X chromosome with respect to yellow and forked (fig. 1*B*).

A map of the sex-linked factors of D. virilis is given in figure 1*A*.[3]

Crossveinless in Drosophila virilis.—Crossveinless in Drosophila virilis was discovered (November, 1917) in the sons of a female containing the factor yellow in one X chromosome and the factors vesiculated, hairy

magenta, and forked in the other. In the offspring of this female (number 1309, table 4*D*), which had been mated by yellow males, it was noted that all the non-vesiculated sons were crossveinless. This indicated that the locus of the new factor was in the X chromosome very near the vesiculated locus, and that the mutation had occurred in the yellow-bearing chromosome or in one of its ancestors. This chromosome had come from yellow stock, and an examination of the stock showed that a large proportion of the yellow flies were crossveinless. The success of the mutation in establishing itself in the crowded conditions of a stock bottle indicated a good viability, and subsequent experiments have shown that the viability is comparable to that of wild-type flies. The excellent viability and the clear-cut nature of the crossveinless character will make it very useful in determining linkage relations in D. virilis.

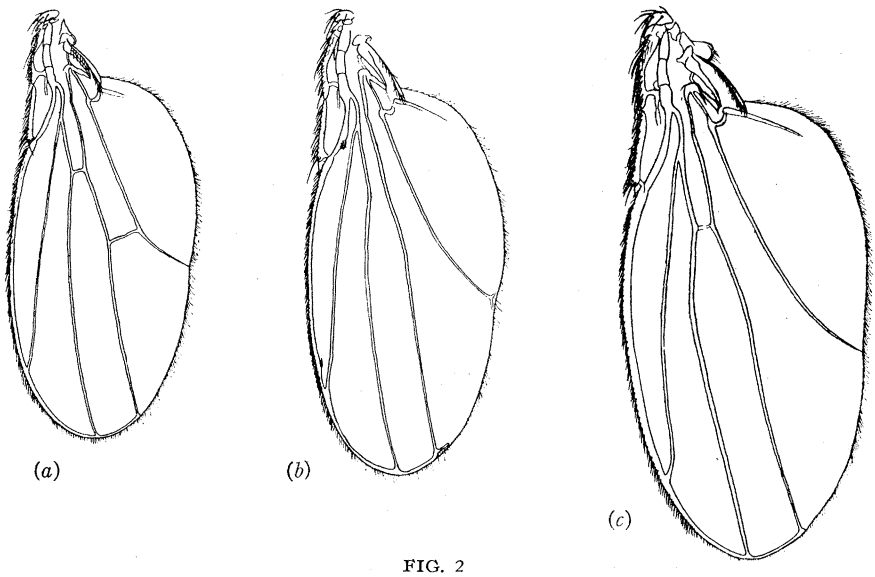

FIG. 2

(*a*) Wild-type wing of D. melanogaster (the wild-type venation of D. virilis is identical with this); (*b*) crossveinless wing of melanogaster; (*c*) crossveinless wing of virilis.

Crosses involving yellow, crossveinless, and vesiculated (table 1) show that the crossveinless locus lies between the other two, very near the vesiculated locus. The yellow crossveinless distance in these experiments was 19.23 units and the crossveinless vesiculated distance 1.15 units. The inclusion of other data (tables 2 and 3) lowers the yellow crossveinless distance to 17.6 units.

The determination of the crossveinless vesiculated distance may be subject to an error due to the fact that vesiculated is not a perfect diagnostic character. Vesiculated flies show the character sometimes in both wings, more often in only one, and occasionally (as we should therefore expect) in neither wing. This last point is proved by the fact that matings

of vesiculated by vesiculated sometimes produce apparently normal flies which, however, behave genetically like vesiculated. Thus an apparently normal female from vesiculated stock mated by a vesiculated male produced 35 vesiculated and 3 normal females, and 48 vesiculated and 1 normal male. It is, therefore, possible that in the present experiment some non-crossover vesiculated flies appeared normal winged and were classed as crossovers between crossveinless and vesiculated. Similarly, some flies that were genetically yellow crossveinless vesiculated may

TABLE 1

OFFSPRING OF $\frac{y\ cv}{vs}$ FEMALES

CULTURE NO.	FEMALES	MALES						
		0		1		2		
		y cv	*vs*	*y vs*	*cv*	*y cv vs*	+	Totals
1591	62	36	29	4	4	1	3	77
1592	90	40	38	9	13		1	101
1593	69	24	29	5	5	1		64
1703	121	37	35	12	7	1		92
1704	113	59	40	9	14		1	123
1705	106	42	34	10	14		1	101
1706	120	42	43	11	9	1	1	107
1707	116	45	46	8	11		1	111
1708	52	24	21	2	1			48
1709	84	19	31	8	6	2		66
1710	88	34	43	7	6			90
1711	93	22	43	12	5			82
1712	91	38	32	11	14		1	96
1713	65	24	23	5	12		1	65
1714	59	12	18	8	1			39
1715	76	40	29	9	5	1		84
1716	88	32	23	7	6			68
1717	74	20	31	4	7			62
1718	91	31	33	7	12	1		84
Totals	1658	621	621	148	152	8	10	1560

have appeared and been classed as yellow crossveinless. This second error would tend to counterbalance the first, but only slightly, because the much larger size of the non-crossover class would make the number of vesiculated flies simulating normal much greater than the number of yellow crossveinless vesiculated flies simulating yellow crossveinless. Consequently, the crossveinless vesiculated distance, if in error, should be shorter than the calculated value. It seems, however, unlikely that in this experiment any large error was introduced in this way, because

TABLE 2

A. OFFSPRING OF $\frac{y\ cv}{m\ f}$ FEMALES

CULTURE NO.	FEMALES	MALES										
		0		1		2		3		1,2		1,3
		y cv	*m f*	*y m f*	*cv*	*y cv m f*	+	*y cv f*	*m*	*y*	*cv m f*	*y m*
1607	69	21	13	1	3	8	7					
1608	34	7	6	2		7	2		1	3		
1611	45	2	10		1	4	5		1		1	
1612	53	11	14			8	10	1				
1613	45	14	13	2	1	6	7			1	2	1
Totals...	246	55	56	5	5	33	31	1	2	4	3	1

B. OFFSPRING OF $\frac{y\ cv\ m\ f}{r}$ FEMALES.

CULTURE NO.	FEMALES	MALES																	
		0		1		2		3	4		1,2		1,3	1,4		2,4		3,4	1,2,3	1,2,4
		y cv m f	*r*	*y r*	*cv m f*	*y cv r*	*m f*	*f*	*y cv m f r*	+	*y m f*	*cv r*	*cv m r*	*y*	*cv m f r*	*y cv*	*m f r*	*y cv m*	*y m r*	*cv*
1658	110	13	14	3	7	10	16		3	8	1	3				4	1	1		
1662	69	4	9	3	1	8	5			5		1		2		2		1		
1663	110	6	21	3	2	10	9			5	1		2	2		2	1	1		1
1670	118	16	20	8	1	8	5		3	4	1	2		3		7	1		1	1
1675	57	11	13	1	4	7	3	1	1	2	2				1	6				
Totals..	464	50	77	18	15	43	38	1	7	24	5	6	2	7	1	21	3	3	1	2

C. OFFSPRING OF $\frac{y\ cv}{m\ f\ r}$ FEMALE

CULTURE NO.	FEMALES	MALES								
		0		1	2	3	4	1,4	2,3	2,4
		y cv	*m f r*	*cv*	+	*m*	*y cv r*	*cv r*	*y cv m*	*r*
1559	36	12	3	2	8	3	3	2	1	1

the complementary classes are so nearly equal. Moreover, if there was overlapping between vesiculated and normal, we should expect some genetically yellow vesiculated flies to appear merely yellow (thus simulating double crossovers); but no merely yellow flies were observed in this cross. Whatever error there may be does not influence the determination of the yellow crossveinless distance; furthermore, the order of the factors is not obscured.

Linkage of Crossveinless with Magenta and Forked.—Crosses of crossveinless with magenta and forked indicate that the crossveinless magenta distance is 36.0 and the magenta forked distance 2.7 units. This would place forked 38.7 units from crossveinless and 56.3 units from yellow. However, the crossveinless magenta distance is so long that double crossing over occurs within it (see tables 3B and 4C); and the discovery of other mutations within this region will probably move magenta and forked farther to the right. If the ratio of the crossveinless forked distances in D. virilis and D. melanogaster is the same as that of the yellow crossveinless regions (17.6 to 13.7), forked in D. virilis should be about 54 units from crossveinless and about 71 units from yellow.

The Locus of Hairy.—Crosses involving crossveinless and hairy (table 3) indicate that the two loci are about 31 units apart.

TABLE 3

A. SONS OF $\frac{y \ cv}{h}$ FEMALES

CULTURE NO.	0		1		2		1,2	
	y cv	*h*	*y h*	*cv*	*y cv h*	+	*y*	*cv h*
1563	15	13	3	2	6	9	1	
1564	6	3				4		1
1567	5	7	1	2		4		
Totals...	26	23	4	4	6	17	1	1

B. SONS OF $\frac{y \ cv \ h \ m}{}$ FEMALES

CULTURE NO.	0		1		2		3		1,2		1,3		2,3	
	y cv h m	+	*y*	*cv h m*	*y cv*	*h m*	*y cv h*	*m*	*y h m*	*cv*	*y m*	*cv h*	*y cv m*	*h*
1422	15	15	5	3	11	4			2	1		1		
1424	19	37	5	2	17	8	5	1		5	1	1	1	1
Totals.....	34	52	10	5	28	12	5	1	2	6	1	2	1	1

The second part of this table suggests that hairy lies to the left of magenta, because when these two factors separate, yellow and crossveinless generally go with hairy. This is contrary to the conclusion of Metz (1918)[1] who provisionally located hairy between magenta and forked on the basis of crosses not involving hairy and magenta simultaneously. The following crosses (table 4), in which all three factors were present at the same time, show clearly that the actual order is hairy, magenta, forked.

TABLE 4

A. OFFSPRING OF $\frac{m\ \ f}{h}$ FEMALES

CULTURE NO.	FEMALES	MALES					
		0		1		2	
		h	*m f*	*h m f*	+	*h f*	*m*
1531	60	52	7		2		
1539	60	16	17	1	2	2	3
Totals.....	120	68	24	1	4	2	3

B. OFFSPRING OF $\frac{y\ \ m\ \ f}{h}$ FEMALE

CULTURE NO.	FEMALES	MALES									
		0		1		2		3	1,2	1,3	
		y m f	*h*	*y h*	*m f*	*y*	*h m f*	*y m*	+	*y h f*	*m*
1352	83	13	24	16	9	1	4	1	2	1	1

C. OFFSPRING OF $\frac{y\ \ \ \ m\ \ f}{vs\ \ h}$ FEMALES

CULTURE NO.	FEMALES	MALES											
		0		1		2		3	4	1,2		1,4	2,3
		y m f	*vs h*	*y vs h*	*m f*	*y h*	*vs m f*	*y*	*vs h f*	*y vs m f*	*h*	*m*	*vs*
1351	33	6	6	3	2	14	2	1			2		
1353	88	8	16	1	2	5		2	1	1	1	2	1
Totals...	121	14	22	4	4	19	2	3	1	1	3	2	1

TABLE 4—(*Continued*)

D. OFFSPRING OF $\frac{y}{vs\ h\ m\ f}$ FEMALE

CULTURE NO.	♀ ♀	MALES																							
		0		1		2		3		4		1,2		1,3		1,4		2,3		2,4		1,2,3	1,2,4	
		y	*vs h m f*	*y vs h m f*	+	*y h m f*	*vs*	*y m f*	*vs h*	*y f*	*vs h m*	*y vs*	*h m f*	*y vs h*	*m f*	*y vs h m*	*f*	*y h*	*vs m f*	*y h m*	*vs f*	*y vs m f*	*y vs f*	*h m*
1305	102	32	4	1	3	6	10					3	1		1		1	1						
1306	59	12	1	3	4	3	6				2	2	1	1		1								
1309	104	29	14		7	11	15	2	1			2	4			1			1					
1335	147	35	9	6	9	8	16	2	3		1	9	3	1			4	1	1					1
1338	98	23	2	1	8	4	5		1	1	1						2							
1340	87	12	1		3	4	7	2									3	1						
1342	64	18	3	1	5	8	14	2		4		1									1			
1343	84	36	6		6	8	17	4	1	1		1	2				2							
1344	76	27	9	1	3	7	14			1	1	2	2				1							
1359	68	24	6	1	4	3	6		1	1			2					2		1				
1360	84	17	6	1	4	5	11	2	2	1		1		3						1				
1361	54	16	3	4	2	4	3					2	1	1						1				
1362	83	13	10	4	4	5	11		1	2	1	3					1			1			1	
1363	53	16	5	4	4	10	9	1				1			1				1			1		
Totals...	1163	310	79	27	66	86	144	15	10	11	6	27	16	6	2	2	14	5	3	4	1	1	1	1

These results demonstrate, despite the great differential mortality in many broods, that the hairy locus is to the left of magenta; because whenever there is crossing over between hairy and magenta, forked goes with the linked gene in the magenta locus, and vesiculated (except for occasional double crossing over) goes with the linked gene in the hairy locus. If we consider only those broods in which there is no marked differential mortality (A1539 and B), we obtain the same result.

Linear Linkage in Drosophila virilis.—The data on the linkage of yellow, crossveinless, and vesiculated, and of hairy, magenta, and forked, are of interest because they demonstrate that in each case the three genes are in strictly linear order, the longest distance being the exact sum of the

TABLE 5

A. OFFSPRING OF $\frac{y}{h\ \ m}$ FEMALE

CULTURE NO.	FEMALES	MALES							
		0		1		2		1,2	TOTALS
		y	*h m*	*y h m*	+	*y m*	*h*	*m*	
1369	89	35	29	23	18	1	4	1	111

B. OFFSPRING OF $\frac{h}{m}$ FEMALE

CULTURE NO.	FEMALES	MALES			
		h	*m*	*h m*	TOTAL
1529	71	22	7	1	30

two shorter distances. Hitherto the only species for which such data have been presented has been D. melanogaster, in which, as Sturtevant, Bridges, and Morgan,[4] and Muller[5] have shown by reference to the results of critical tests, the arrangement of the genes is exactly linear. As a result of criticisms by these authors, Castle[6] has withdrawn his objections to the theory of linear linkage. In D. virilis, however, there have been hitherto no decisive data published. Metz's results, while they agree so far as they go with those in melanogaster, do not go far enough to give a decisive proof of linear arrangement. In the only case (that of hairy, magenta, and forked) in which the factors with which he worked were sufficiently close together to make a crucial test possible, he failed to make the necessary cross.

Castle, on the basis of his three dimensional theory and of Metz's data, arranged hairy, magenta, and forked, at the apices of a triangle, and predicted that the hairy magenta distance (which Metz had not determined) would be "about 4 or 5." The data now presented (tables 3 B, 4, and 5) give the hairy magenta distance as 5.5 units; or—if only those cultures are used in which the viability is good (tables 3 B; 4 A 1539, B; 5A)—the hairy magenta distance is 7.03 units.

Of course, because of the poor viability in the one case and the small numbers in the other, little reliance can be placed on these determinations as exact values. However (and this is the point at issue) the uncertainty does not affect the arrangement of the loci, which is clearly a linear one; for in all the experiments where the three loci were followed simultaneously, the frequency of separation of hairy and forked (0.1003) was the exact sum of the hairy magenta (0.0534) and the magenta forked (0.0469) separation frequencies.

Now the linear order, besides disproving Castle's triangular arrangement, shows that the values he used were not consistent with each other; for the hairy forked distance, being necessarily longer than the magenta forked distance, cannot be 3.1 if magenta forked is 3.7. The two distances, not being strictly comparable, should not be used to predict the hairy magenta distance definitely. Moreover, if the hairy magenta distance is (as Castle predicts) 4 or 5, the hairy forked distance must be longer than 4 or 5 and cannot be 3.1; that is, if Castle's predicted value is correct it follows that the value on which the prediction was based is incorrect. Thus the fulfilment of the prediction would in itself invalidate the grounds on which the prediction was based. It is therefore of no consequence whether or not the hairy magenta value recorded in this paper (5.5 for the total broods or 7.03 for those with good viability) be considered in agreement with Castle's "about 4 or 5 units"; for any agreement must be accidental and the fulfilment or non-fulfilment of Castle's prediction can have only a dramatic and not a scientific value.[7]

The loss of the hairy stock prevents any further determinations; but the data already obtained are sufficient to demonstrate that the hairy, magenta, and forked loci are arranged in strictly linear order. It has already been noted that in the cross involving simultaneously yellow, crossveinless and vesiculated, the yellow vesiculated separation frequency (20.38) is the exact sum of the yellow crossveinless (19.23) and the crossveinless vesiculated (1.15) separation frequencies. Hence, in the two only cases in D. virilis in which (because of the absence of double crossing over) the linear theory can be subjected to a decisive test, the arrangement of the genes in the chromosome turns out to be precisely linear.[8]

Castle also predicted, on the basis of his theory, that rugose and glazed would give a crossing over value of about 4 or 5 percent.; but this prediction cannot be tested directly because rugose-glazed hybrids are

sterile. It is, of course, possible to determine the crossing over value of each gene with the nearest other factor and to compare the distances thus obtained. But such an experiment is necessarily inconclusive because variations in linkage caused by other genetic factors are difficult or impossible to eliminate unless the loci to be compared are followed in the same cross.

Castle's prediction can, however, be disproved in another way, because there is evidence that rugose and glazed are allelomorphs. This is indicated by the similarity of their linkage relations (so far as tested) and of their somatic effects. Rugose dominates glazed, but this cannot be considered evidence of allelomorphism because rugose females, and hence rugose-glazed hybrid females, are wild-type in appearance. (As the factors are sex-linked there are, of course, no males carrying both together.) However, I have found that glazed females are almost invariably sterile. (Out of 146 females tested in my experiments, either singly or in mass cultures, only two produced offspring.) The sterility of glazed, while it is recessive to wild-type fertility, is dominant to the fertility of rugose, so that rugose-glazed hybrid females are sterile. This suggests strongly that the rugose and glazed genes are allelomorphic; and the evidence is strengthened by the behavior of a third mutant gene, wax, which in its somatic effects resembles rugose and glazed (though it is more extreme) and is, like them, sex-limited. Wax females, I have found, are also sterile. (Of 89 tested in my experiments, all but two failed to give offspring.) Wax eye is recessive to wild-type and to rugose, and the sterility of wax is recessive to the fertility of wild-type, but dominant to the fertility of rugose. (None of the 84 rugose-wax females tested in my experiments produced offspring.) Wax also, therefore, seems to be allelomorphic to rugose, and this is confirmed by the fact that glazed and wax behave as allelomorphs when crossed: the hybrid is intermediate and, as would be expected, sterile. (None of the 106 tested produced offspring.)[9]

Since the evidence indicates that rugose and glazed are allelomorphs, the amount of crossing over between them, if it could be directly determined, would be not about 4 or 5 per cent (as Castle predicted), but 0.

Coincidence in Drosophila virilis.—While the data in Drosophila virilis are not sufficiently extensive for any detailed considerations of coincidence, they indicate, as far as they go, that the coincidence of various regions resembles that of the corresponding regions of the X chromosome in D. melanogaster.

The values of table 6, because of the smallness of the figures and the uncertainty of vesiculated as a diagnostic character, are subject to large error; but they all agree in indicating that coincidence first

TABLE 6

COINCIDENCE IN D. VIRILIS

REGIONS	COINCIDENCE	SOURCE OF DATA
Yellow crossveinless and crossveinless vesiculated	0	This paper, table 1
Yellow crossveinless and crossveinless magenta	0.848	This paper, tables 2, 3B
Yellow vesiculated and vesiculated magenta	0.977	Metz, 1918, tables 11, 19, 22
Yellow crossveinless and magenta forked	1.4	This paper, table 2
Yellow vesiculated and magenta forked	0.989	Metz, 1918 table 19
Yellow crossveinless and forked rugose	0.795	This paper, tables 2B, C
Yellow vesiculated and forked rugose or forked glazed	0.590	Metz, 1918 tables 20, 21

increases and then decreases as the regions considered become separated farther. This agrees very closely with the conditions shown to exist in the X chromosome of D. melanogaster by Muller (1916)[10] and Weinstein (1918).[2] A similar rise and fall of coincidence have been demonstrated for the second chromosome of D. melanogaster by Bridges[2] and for the third chromosome by Gowen (1919).[11]

The Homology of Apparently Similar Factors.—Although the evidence so far obtained favors the homology of the yellow crossveinless forked series in D. virilis and D. melanogaster, it should be kept in mind that the data are by no means sufficient to make a homology certain for it is known that similar somatic effects may be produced by non-homologous genes. Thus in melanogaster there are mutant characters that resemble each other in appearance but are due to mutant genes in different loci; for example, the various eye colors resembling pink (ruby, garnet, purple, maroon, claret, etc.) and the various body colors resembling black (sable, ebony). In some cases two such "mimic" genes have been found to be very close together in the same chromosome, as in the case of miniature and dusky, which are not more than 1.8 units apart. It is obvious that a miniature mutation in virilis, even if it showed closely similar linkage relations, could not be definitely homologized with either of these two genes. A yellow virilis male with miniature wings did, indeed, occur in a cross of a yellow female by a wild-type male; and this miniature variation, if a mutation and recessive, must have been sex-linked. Unfortunately it proved sterile and its linkage relations could not be tested. Since mutations that have similar somatic effects may occur in homologous chromosomes of the same species and occupy non-homologous loci, it is certainly hazardous to take it for granted that mutations of similar effect

occurring in homologous chromosomes of different species must lie in homologous loci.

A case particularly in point in the present connection is that of the body color chlorotic, resembling yellow; its gene is close to the yellow gene in the melanogaster X chromosome. Another case is that of magenta in virilis. If forked were not known in this species, magenta might have been homologized with garnet, which is twelve units to the left of forked in melanogaster. While this identification is not entirely impossible, it is rendered rather improbable by the difference in distance from forked and by the large number of magenta-like mutants in melanogaster. The cases of singed and inflated have already been considered.

Not only may similar effects be caused by non-homologous genes, but two originally identical genes may, because of the mutation of one, or the mutation of both in different directions, or because of differences in modifying factors, come to have different somatic effects. Modifying factors may also affect the strength of linkage. The differences between crossveinless in melanogaster and virilis, and the differences between the lengths of the yellow crossveinless and the crossveinless forked distances in the two species, might be thus explained.

The order of the genes would not be affected by modifying factors (though if crossing over were entirely prevented, the order would be impossible to determine, at least in the ordinary way). But Bridges's cases of duplication and transposition[12] show that genes may be shifted to another region of the chromosome, or even to a different chromosome, without being otherwise affected; so that homologous (and indeed apparently identical) genes may come to have different linkage relations. By a combination of mutation and transposition, homologous factors might come to differ in both chemical composition and in linkage relations.

Further complications might be introduced because of duplication and of deficiency. In the vermilion duplication stock of melanogaster, each X chromosome carries two vermilion genes. Though one vermilion gene is recessive to wild-type, two vermilion genes dominate one not-vermilion. If a low cross-over factor were introduced, the two vermilion genes would remain together and would simulate the behavior of a single dominant gene. If vermilion-deficiency were substituted for one vermilion gene, the vermilion vermilion-deficiency combination would simulate a dominant vermilion with a recessive lethal effect.[13]

A similar reversal of dominance might occur because of the presence of several recessive genes in tetraploidy. Such a case could be distinguished by the frequencies of the different classes, for each factor would act as an allelomorph to each of the others.[14] Finally, there might also be reversal of dominance because of modifying factors.[15]

It must therefore be understood that while the discovery of crossveinless in D. virilis and D. melanogaster increases the probability that

the X chromosomes of the two species are composed of homologous genes, a demonstration of such homology in the absence of hybridizing experiments requires a greater number of similar genes similarly placed than have yet appeared.

Are Genes with Similar Somatic Effects Chemically Similar?—It might be supposed that factors with similar somatic effects are chemically similar even if they occupy different loci. But this supposition can be disproved by a consideration of two such factors as ruby and garnet in D. melanogaster. Each of these produces a pink eye, but the genes lie in different parts of the X chromosome. A ruby female has the composition rG/rG, a garnet female the composition Rg/Rg. Now, if $r = g$ and $R = G$, either of the above genetic compositions should produce the same effect as the composition rg/RG or rG/Rg; for in each of these cases there are two (supposedly similar) genes for pink eye color (r and g) and two (supposedly similar) allelomorphs of these (R and G). But the composition rg/RG or rG/Rg produces not a pink but a red (wild-type) eye. Hence either r differs from g, or R differ from G, or both.

This proof depends on the assumption that the position of a gene does not influence its somatic expression. The correctness of this assumption is demonstrated by Bridges'[11] cases of duplication and transposition already referred to, in which the shifting of a factor from one part of a chromosome to another or even to a different chromosome does not alter its somatic effect.

[1] Hyde, R. R., *Amer. Nat.*, **49**, 1915 (183–85, 185–187); Metz, C. W., *Genetics*, **1**, 1916 (591–607); *Ibid.*, **3**, 1918 (107–34); Sturtevant, A. H., *Science*, **48**, 1918 (72–3).

[2] Weinstein, A., *Genetics*, **3**, 1918 (135–73).

[3] D. virilis itself has been described by Sturtevant, *Ann. Ent. Soc. Amer.*, **9**, 1916 (323–43). Descriptions of the sex-linked mutants other than crossveinless will be found in Metz, *l. c.* Dscriptions of the sex-linked characters in D. melanogaster will be found in Morgan and Bridges, *Carnegie Institution Washington, Publ.*, No. 237, 1916 (1–87).

[4] Sturtevant, A. H., Bridges, C. B., Morgan, T. H., These PROCEEDINGS, **5**, 1919 (168–173); Morgan, T. H., Sturtevant, A. H., and Bridges, C. B., *Ibid.*, **6**, 1920 (162–164).

[5] Muller, H. J., *Amer. Nat.*, **54**, 1920 (97–121).

[6] Castle, W. E., These PROCEEDINGS, **5**, 1919 (25–32, 32–36); *Ibid.*, **5**, 1919 (500–506); *Ibid.*, **6**, 1920 (73–77).

[7] It was pointed out in Muller's article that the value which I had recently obtained for the hairy magenta distance was (on the data then available) 6.6, contrary to Castle's prediction; and that this, if combined according to Castle's own method with the values previously obtained by Metz in separate crosses for the hairy forked and forked magenta distances, would place the three factors in a nearly straight line in the order hairy forked magenta. This situation was used by Muller to show that, even by using Castle's method of combining separate experiments, linear results might be arrived at that were quite inconsistent with the prediction obtained by the same method. The new value, moreover, was based on more flies (total 364) than Metz's hairy forked value used by Castle (162). Muller made no claim, however, that these relationships were really correct, as it was evident that linear results arrived at by such a method

might be accidental, just as Castle's non-linear results so obtained were accidental. The fact that the order really turns out to be hairy magenta forked illustrates the latter point to a nicety and falls in line with the other results to show the fallacy of the method of combining separate experiments for a determination of the nature of linkage.

[8] It is of course possible that larger counts would give double crossovers in these cases. But this is very improbable in the case of hairy, magenta, and forked, because of the shortness of the distance involved; and even in the case of yellow, crossveinless, and vesiculated, where the distance is longer, there could scarcely be enough double crossing over to throw the factors much out of line.

[9] The sterility of rugose-glazed hybrids was first observed by Metz, who attributed it to "incompatibility," because he thought that glazed females were fertile. He had noticed their sterility when mated to rugose males, but he termed this also "incompatibility," as he believed that glazed females were not sterile when mated to glazed or to wild-type males. Now that it is known that glazed females are almost always sterile, it is obvious that the failure of the hybrids to produce offspring is due to the dominance of the sterility of glazed. In my work, each class of females (glazed, wax, and hybrids) was tested with four kinds of males (wild-type, rugose, glazed, and wax); and in all cases, with the exceptions already noted, the matings produced no offspring, though all four types of males are otherwise fertile. Metz has, consequently, withdrawn his "incompatibility" theory and accepted the interpretation given above.

[10] Muller, H. J., *Amer. Nat.*, **50,** 1916 (193–221, 284–305, 350–366, 421–434).

[11] Gowen, J. W., *Genetics*, **4,** 1919 (205–250).

[12] Bridges, C. B., *Genetics*, **2,** 1917 (445–465); *J. Gen. Physiol.*, **1,** 1919 (645–656).

[13] The yellow genes in mice and rats suggest themselves in this connection; but there is no proof that the yellow mouse case is actually like the hypothetical vermilion vermilion-deficiency combination described above, or that yellow in mice is homologous with either of the yellow factors in rats.

[14] Muller, H. J., *Amer. Nat.*, **48,** 1914 (508–512).

[15] Altenburg, E., and Muller, H. J., *Genetics*, **3,** 1920 (1–59); Bridges, C. B., and Mohr, O. L., *Genetics*, **4,** 1919 (283–306).

THE INTENSITIES OF X-RAYS OF THE L-SERIES, III. CRITICAL POTENTIALS OF THE PLATINUM AND TUNGSTEN LINES

BY FRANK C. HOYT

ROGERS LABORATORY OF PHYSICS, MASSACHUSETTS INSTITUTE OF TECHNOLOGY

Communicated by Edwin B. Wilson, September 27, 1920

This work is a continuation of that of Webster and Clark[1] and Webster.[2] The methods used by them are applied to the classification of some lines that were doubtful, and the general validity of the intensity laws is tested by the use of a different metal as anticathode (tungsten).

Apparatus.—The high tension outfit constructed at the Massachusetts Institute of Technology by Prof. D. L. Webster and described in these PROCEEDINGS[3] was used without essential modification. The Chaffee